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CASE REPORT

Echocardiographic progression of a subepicardial aneurysm after inferior myocardial infarction

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Summary A 62-year-old man presented to hospital with chest oppression. Coronary angiography revealed total occlusion of the right coronary artery and inferior myocardial infarction was diagnosed. He was treated by percutaneous coronary intervention with stenting for myocardial infarction. After four months, echocardiography revealed a huge aneurysm protruding below the inferior surface of the left ventricle. It was considered to be a pseudoaneurysm from the echocardiographic findings. The patient had no symptoms and he refused surgery, so progression of the aneurysm was monitored carefully. At seven months after revascularization, the aneurysm showed a marked increase in size, with a maximal diameter of 48 mm and orifice diameter of 22 mm. Accordingly, the patient agreed to undergo surgical excision. The aneurysm was resected and the defect was closed with a pericardial patch. Pathological examination revealed all of the myocardial elements in the aneurysm wall and thrombus in its lumen. In conclusion, this was a rare case of venricular subepicardial aneurysm.

Introduction

Subepicardial aneurysm is a rare complication of myocardial infarction. Subepicardial aneurysms of the left ventricle usually feature sudden interruption of the myocardium, a narrow neck, and a

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propensity to rupture spontaneously, regardless of the components of the aneurysm wall. Pathological examination can distinguish true ventricular aneurysms from pseudoaneurysms. However, a subepicardial aneurysm is simply diagnosed from its morphologic features. The most important point is that subepicardial aneurysms tend to rupture and cause sudden death, even in the chronic phase and regardless of their size. Accordingly, these aneurysms are sometimes diagnosed at postmortem examination [1]. When a subepicardial aneurysm is found, surgery should be performed as soon as possible.

A subepicardial aneurysm develops at the site of partial rupture of the infarcted ventricular wall. While the epicardium (sometimes with myocardial remnants) prevents complete ventricular rupture, it is expanded by the high left ventricular pressure to form this unique type of aneurysm [2].

Case report

A 62-year-old man suddenly developed chest pain while walking in February 2006. This symptom continued for about 40 h, but he ignored it until the pain became worse and he could not tolerate it any further. He was then brought to our hospital by ambulance.

On admission, he had a heart rate of 82 bpm and a blood pressure of 111/67 mmHg. The 12-lead electrocardiogram showed inverted T waves in leads II, III, aVf, V4, V5, and V6 along with abnormal Q waves in leads III and aVf. Laboratory tests revealed that serum total cholesterol was 210 mg/dl, triglycerides were 45 mg/dl, low-density lipoprotein was 159 mg/dl, high-density lipoprotein was 42 mg/dl, uric acid was 4.5 mg/dl, and hemoglobin A_{1c} was 7.1%. Transthoracic echocardiography revealed akinesis of the inferior wall of the left ventricle. The infarction area remained in the myocardium (Figure 1A and B). We diagnosed inferior acute myocardial infarction, and emergency cardiac catheterization was performed. Heparin was administered (100 U/kg). Emergency coronary angiography (CAG) showed total occlusion of the right coronary artery (RCA). We performed percutaneous coronary intervention using a 7Fr JR4 guide catheter (Launcher®; Medtronic Ltd., Minnesota, MN, USA) and guide wire (Runthrough®, Terumo Co. Ltd., Tokyo, Japan) to cross the culprit lesion in the RCA. We then performed stenting of the culprit lesion. TIMI grade 2 flow was achieved and the vessel wall distal to the lesion was thin.

After that, his chest symptoms were resolved. We administered nicorandil (0.5 γ) and heparin (400 U/h) by continuous intravenous infusion. His peak serum creatinine kinase level after coronary intervention was 4088 IU/l. He was switched to oral medication with aspirin (100 mg/day) and ticlopidine (200 mg/day). After 7 days, chest roentgenography showed slight cardiomegaly and echocardiography revealed a moderate pericardial effusion. However, there was no abnormal flow from the left ventricle to the epicardium. Echocardiography revealed almost the same view as on admission, except pericardial effusion. He had no symptoms and his condition was stable with a heart rate of 92 bpm and a blood pressure of 110/70 mmHg, so we administered diuretics and observed him carefully. The pericardial effusion gradually decreased in volume and his general condition remained good. Follow up CAG revealed patency of the stent and the patient was discharged from hospital on March 26.

Four months later, two-dimensional echocardiography revealed a large anechoic cavity behind the inferior wall of the left ventricle that communicated with the left ventricular lumen (Figure 1C). The aneurysm of inferior wall of the left ventricle was sudden interruption of the myocardium. Blood flowed from the left ventricle into the cavity during systole and flowed in the opposite direction during diastole on color Doppler imaging. The maximum diameter of the lesion was 28 mm. We concluded that this was a pseudoaneurysm and recommended urgent surgical resection. However, the patient had no symptoms, so he rejected surgery.

After one month, echocardiography revealed expansion of the aneurysm (Figure 1D), with the maximal diameter increasing to 32 mm.

After two months, echocardiography revealed rapid expansion of the aneurysm (Figure 1E). Its maximal diameter was now 48 mm and the orifice measured 22 mm. Multidetector row computed tomography (MDCT) showed a huge aneurysm lying below the inferior surface of the left ventricle (Figure 2A and B). Blood flow from the left ventricle into the cavity during systole and reverse flow during diastole was observed more clearly than that by echocardiography. The walls of the aneurysm were thin, and the myocardium showed sudden discontinuity at the neck of the aneurysm. We feared that the aneurysm would rupture or would cause thrombosis or a ventricular arrhythmia, so we recommended immediate resection and the patient agreed at this time.

On November 20, ventriculoplasty was performed. There was a huge aneurysm on the inferior surface of the left ventricle with epi-

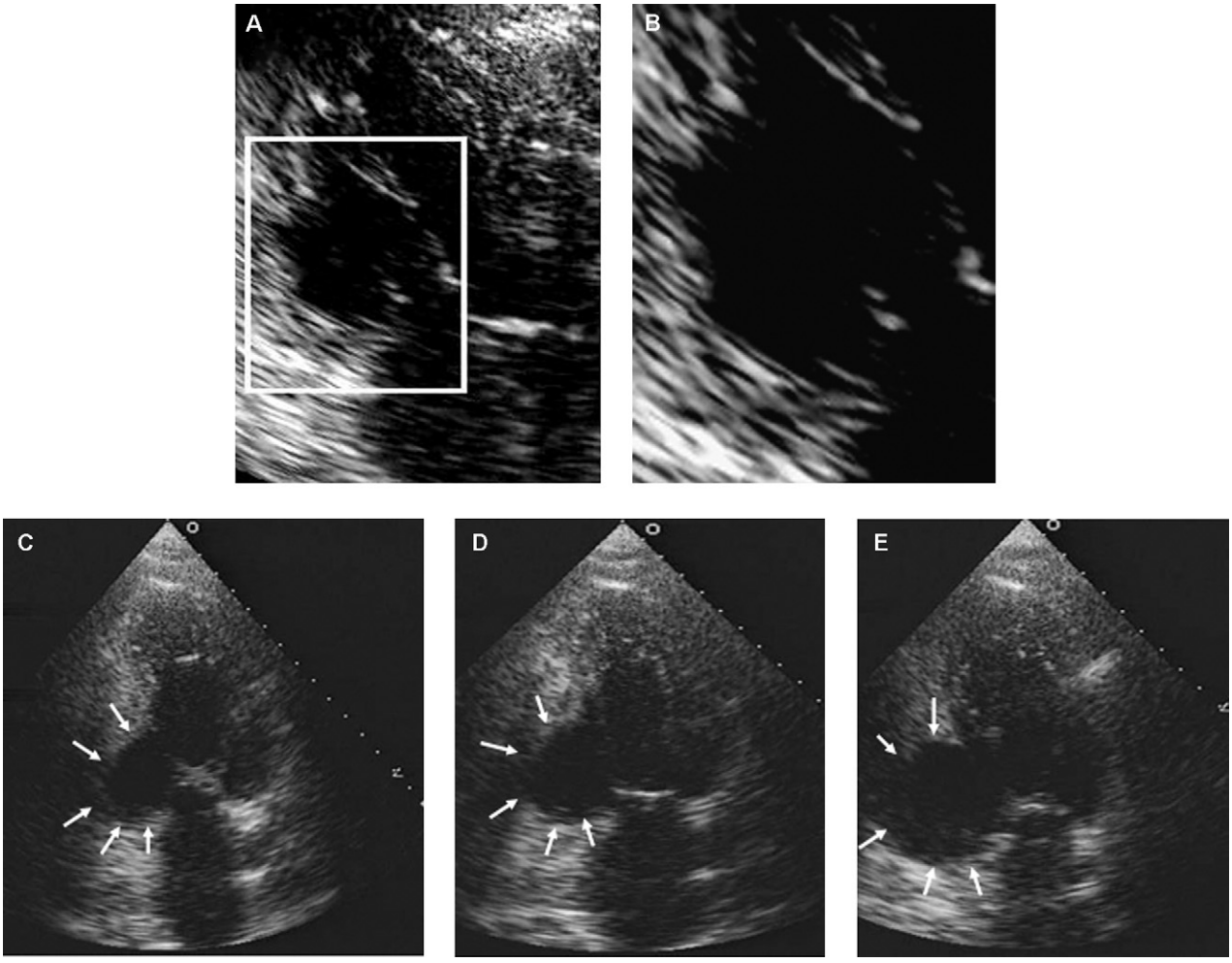

Figure 1 (A) Echocardiography showed akinesis of the inferior wall of the left ventricle on admission. (B) The enlargement of the inferior wall. The infarction area remained in the myocardium. (C) At four months after discharge, echocardiography revealed an aneurysm behind the inferior wall of the left ventricle. The maximal diameter of the aneurysm was 28 mm (arrows). (D) After one month more, the aneurysm was larger and its maximal diameter was 32 mm (arrows). (E) After two months more, there was rapid expansion of the aneurysm and the maximal diameter was 48 mm (arrows).

cardium adherent to its walls. The aneurysm was excised and the defect was closed with a pericardial patch. Pathologic examination revealed the complete myocardium within the aneurysm wall and thrombus in the cavity (Figure 3). Postoperative echocardiography and MDCT showed that the aneurysm was no longer detectable and abnormal blood flow had disappeared. His postoperative course was uneventful.

Discussion

Mechanical complications of myocardial infarction are less frequently encountered in the current era of early revascularization [3]. However, these can still be catastrophic complications, especially ventricular free wall rupture.

Pathological examination is needed to distinguish a true ventricular aneurysm from a pseudoaneurysm after myocardial infarction. The former type has myocardium in its wall and the latter does not. Epstein and Hutchins first advocated the concept of subepicardial aneurysm [2]. A typical subepicardial aneurysm of the left ventricle arises at the site of sudden discontinuity of the myocardium, has a narrow neck, and shows a propensity to rupture spontaneously regardless of the mural components. It is diagnosed from morphologic findings. It is important to remember that subepicardial aneurysm can cause sudden death, even though this is a rare complication

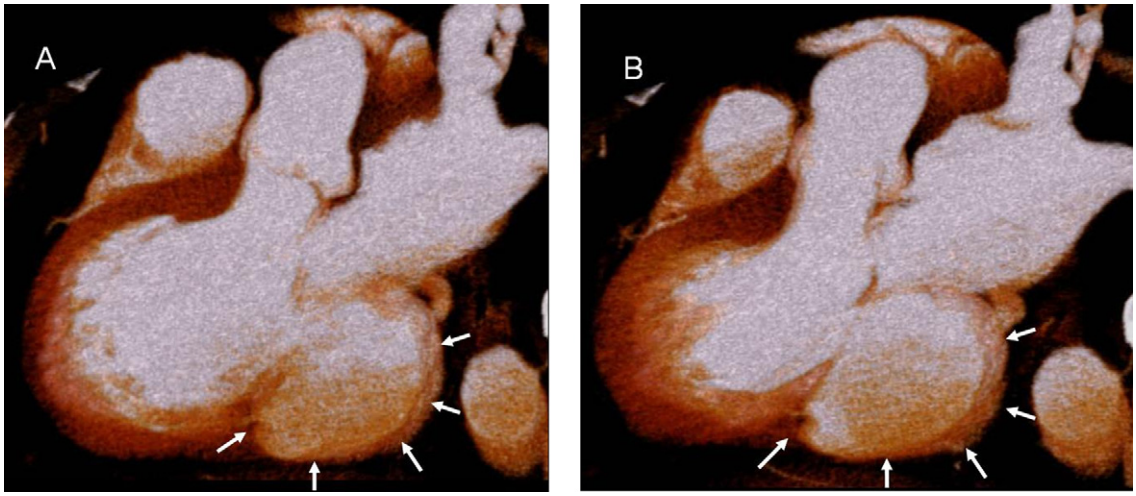

Figure 2 (A) Multidetector row computed tomography shows a huge aneurysm behind the inferior wall of the left ventricle. Blood flows from the aneurysm into the left ventricle during diastole (arrow). (B) Blood flows from the left ventricle into the aneurysm during systole (arrow).

of myocardial infarction. Echocardiography is the method for diagnosing this type of aneurysm [4].

In our patient, a pericardial effusion was detected in the subacute phase of acute myocardial infarction. We suspected that this was due to oozing left ventricular rupture, but the patient's condition was stable and echocardiography did not reveal abnormal flow from the left ventricle. After treatment with a diuretic and observation for several days, his pericardial effusion resolved. This suggested that partial rupture of the infarcted ventricular wall had occurred, which formed a subepicardial aneurysm at four months after his discharge from hospital. The aneurysm enlarged markedly after another three months of observation. This is the first report about serial findings of a subepicardial aneurysm observed by echocardiography.

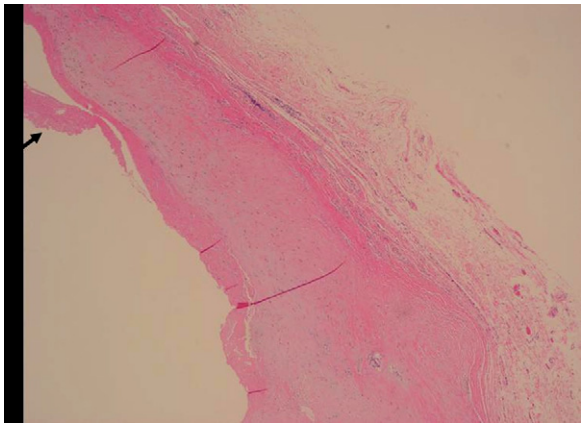

Figure 3 Pathologic examination revealed all of the myocardial elements in the aneurysm wall and thrombus in the lumen of the aneurysm (arrow).

There have been reports that hemorrhagic infarction caused by thrombolysis is associated with incomplete myocardial rupture [5]. Friedman et al. reported that early treatment with aspirin, heparin, and beta-blockers after myocardial infarction may limit infarct size, thereby reducing the risk of infarct expansion and aneurysm formation. On the other hand, steroids, non-steroidal anti-inflammatory drugs, and hypertension may promote aneurysm formation [6]. Our patient was administered aspirin and an angiotensin-converting enzyme inhibitor soon after diagnosis, but an aneurysm formed. He was referred to our hospital about 40 h after infarction occurred, so late revascularization may explain the development of a ventricular aneurysm. Dzavik et al. reported that late revascularization did not reduce infarct size or improve left ventricular function [7]. However, our patient had persistent chest symptoms, so we performed revascularization. Furthermore, we could not achieve TIMI 3 flow. Murakami et al. reported TIMI grade was an important factor for salvaging myocardium in patients with acute myocardial infarction [8]. So, it may be one of the reasons for forming subepicardial aneurysm.

Two-dimensional echocardiography is generally considered to be the technique of choice for diagnosing subepicardial aneurysm. Giltner et al. reported a case of subepicardial aneurysm diagnosed by computed tomography [9]. In our case, MDCT was useful for diagnosis and localization of the aneurysm.

In conclusion, the clinical significance of a subepicardial aneurysm is a high risk of rupture even in the chronic phase and regardless of its size. Accordingly, prophylactic surgical correction is necessary and is usually effective [10].

References

[1] Balakumaran K, Verbaam CJ, Essed CE, Nanta J, Bos E, Haalebos MM, Penn O, Simoons ML, Hugenholtz PG. Ventricular free wall rupture: sudden subacute, slow, sealed and stabilized varieties. Eur Heart 1984;5:282—8.

[2] Epstein JI, Hutchins GM. Subepicardial aneurysm: a rare complication of myocardial infarction. Am J Med 1983;75:639—44.

[3] Pollak H, Nobis H, Mlczoch J. Frequency of left ventricular free wall rupture complicating acute myocardial infarction since the advent of thrombolysis. Am J Cardiol 1994;127:212—4.

[4] Shiraishi H, Urao N, Tsukamoto M, Hyogo M, Keira N, Hirasaki S, Ishibashi K, Shirayama T, Nakagawa M. Subepicardial aneurysm associated with ventricular septal perforation showing a normal coronary angiogram. Circ J 2003;67:962—4.

[5] Gatewood Jr RP, Nanda NC. Differentiation of left ventricular pseudoaneurysm from true aneurysm with two dimensional echocardiography. Am J Cardiol 1980;46: 869—78.

[6] Friedman BM, Dunn MI. Postinfarction ventricular aneurysms. Clin Cardiol 1995;18:505—11.

[7] Dzavik V, Buller CE, Lamas GA, Rankin JM, Mancini GB, Cantor WG, Carere RJ, Ross JR, Atchison D, Forman S, Thomas B, Buszman P, Vozzi C, Glanz A, Cohen EA, et al. Randomized trial of percutaneous coronary intervention for subacute infarct-related coronary artery occlusion to achieve long-term patency and improve ventricular function: the Total Occlusion Study of Canada (TOSCA)-2 trial. Circulation 2006;114:2449—57.

[8] Murakami J, Toyama T, Adachi H, Hoshizaki H, Oshima S, Kurabayashi M. Important factors for salvaging myocardium in patients with acute myocardial infarction. J Cardiol 2008;52:269—75.

[9] Giltner A, Marelli D, Halpern E, Savage M. Subepicardial aneurysm with impending cardiac rupture: a case of antemortem diagnosis and review of the literature. Clin Cardiol 2007;30:44—7.

[10] Stewart S, Huddle R, Stuard I, Schreiner BF, De Weese JA. False aneurysm and pseudo-false aneurysm of the left ventricle: etiology, pathology, diagnosis, and operative management. Ann Thorac Surg 1981;31:259—65.